CASE REPORT

Delayed cerebral vasospasm following traumatic acute subdural hematoma: case report

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Abstract

Introduction: Post-traumatic vasospasm is a rare but known ischemic damage after severe traumatic brain injury that independently predicts patients' outcome. Although the pathogenesis and risk factors have not been elucidated, some reports describe relationship between the occurrence of vasospasm and traumatic subarachnoid hemorrhage. Here, we report a case of vasospasm in a patient with acute subdural hematoma in which traumatic subarachnoid hemorrhage was not recognized both surgically and radiologically.

Case report: A 60-year-old male was admitted for head trauma. Neurologically, he was somnolence and showed right-sided hemiparesis. Computerized tomography (CT) revealed large acute subdural hematoma in the left side associated with midline-shift. He underwent urgent craniotomy and hematoma evacuation. Postoperatively, he recovered well with resolution of neurological symptoms. Follow-up CT revealed complete removal of hematoma. However, his level of neurological status deteriorated on the 5th day after surgery. CT excluded ischemic lesion, but 3D-CT angiography revealed diffuse vasospasm in the left middle cerebral artery, and perfusion imaging confirmed a zone of altered cerebral blood flow in left frontotemporal region. Subsequently, his neurological condition recovered gradually and he was discharged ambulatory on the 9th day after the surgery. Follow-up angiography showed the spasm had disappeared completely.

Conclusion: Post-traumatic vasospasm without traumatic SAH was described. The etiology and pathogenesis of this fairly rare condition associated with head trauma is discussed.

Keywords: cerebral vasospasm; traumatic brain injury; subdural hematoma; delayed cerebral ischemia

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Introduction

Cerebral vasospasm is an arterial narrowing, usually following the subarachnoid and/or intraventricular hemorrhages in course of the aneurysm rupture [1]. Moderate or severe vasospasm develops in roughly two thirds, followed by the delayed cerebral ischemia in one third, followed by infarction in one sixth of patients with subarachnoid hemorrhage (SAH).

Post-traumatic vasospasm (PTV) is not common but it is a well-known complication related to the severe traumatic brain injury that independently comprises the outcomes thorough a delayed cerebral ischemia mechanism [2].

Although the pathogenesis and risk factors have not been elucidated, there are several current pathophysiological mechanisms based on the molecular level response, underlying the occurrence of the PTV, while the mechanical theories based on the direct stretching were abandoned [3]. These theories are all focused to describe this occurrence considering the presence of traumatic SAH.

Conversely, pure acute subdural hematoma of traumatic or spontaneous origin is considered to be a rare cause of the PTV [4]. We report a case of a delayed PTV after the surgery for traumatic acute subdural hematoma (ASDH) in which traumatic SAH was excluded both intraoperatively and radiologically.

Case report

A 60-year-old male was referred from the local hospital after a head trauma, and CT confirmed ASDH. On admission, the patient was somnolent - Glasgow coma scale (GCS): 12 (E2V4M6), with equal pupils and prompt light reflex, and showed mild right-sided hemiparesis. Computerized tomography (CT) revealed enlarged ASDH on the left side associated with moderate midline-shift compared to initial imaging. Traumatic SAH was not evident (***Figure 1A***). Three-dimensional CT angiography (3D-CTA) excluded underlying vascular pathology and cerebral vasospasm (***Figure 2A***).

The patient soon deteriorated neurologically and underwent urgent craniotomy and hematoma evacuation. Intraoperatively, Skull fracture or dural injury were not observed. The hematoma was localized entirely in the subdural space and SAH was not identified in subarachnoid space, basal cisterns or Sylvian fissure. Small laceration of cortical middle cerebral artery branch on the convexity was found to be the bleeding source, and it was completely coagulated. Postoperatively, he recovered well with resolution of neurological symptoms, and CT confirmed complete removal of ASDH (***Figure 1B***).

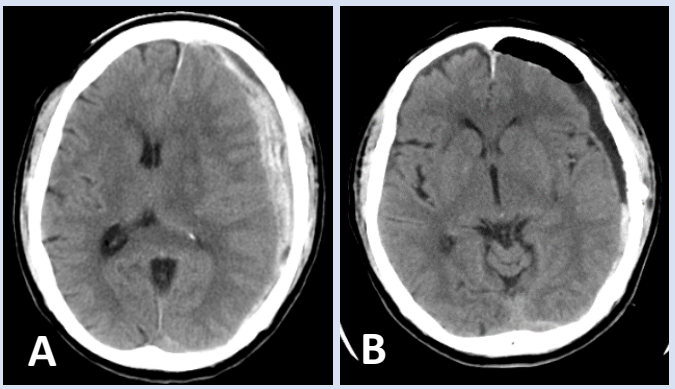

Figure 1. Computerized tomography A. Preoperative, showing left-sided ASDH without traumatic SAH. B. Postoperative, showing complete evacuation of the ASDH with decrease of the mid-line shift.

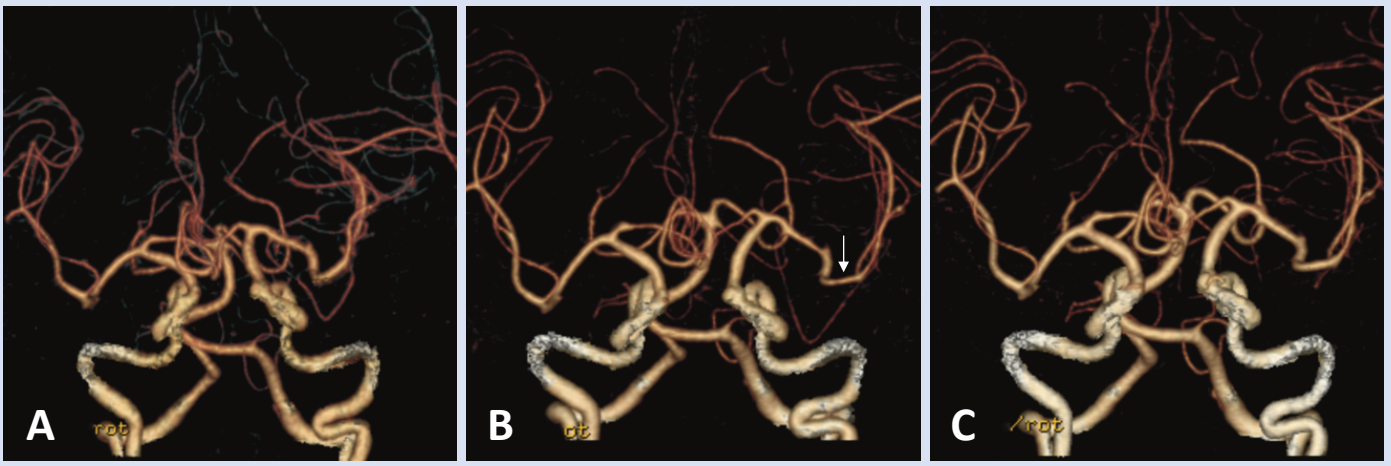

Figure 2. 3D CT angiography findings A. Initial angiography on day 0 showing no vasospasm; B. The arrow points at the vasospasm site on day 5; C. The vasospasm is relieved completely on day 9.

On the 5th day after surgery, the patients' neurological status started to deteriorate with initial dysarthria, which worsened to aphasia within several hours, followed by the development of the right-sided severe hemiparesis. A perfusion CT revealed signs decreased cerebral blood flow (CBF) in the left frontotemporal region, and 3D-CTA found vasospasm of the left sided middle cerebral artery stem (***Figure 2B and 3A***). Vasospasm treatment protocol was introduced with triple H therapy (hypertension, hypervolemia, and hemodilution), with only Atorvastatin 10mg/day as complete vasospasm treatment protocol was contraindicated due to the trauma.

The patient improved gradually over the following days. Hemiplegia improved on the following day, and completely resolved in two days, while the aphasia persisted for 3 days. On the 9th day after the surgery, the patient was discharged ambulatory, with complete symptoms resolution, and 3D-CTA confirmed complete resolution of the vasospasm of the left MCA, while the perfusion CT confirmed blood flow normalized (***Figure 2C and 3B***).

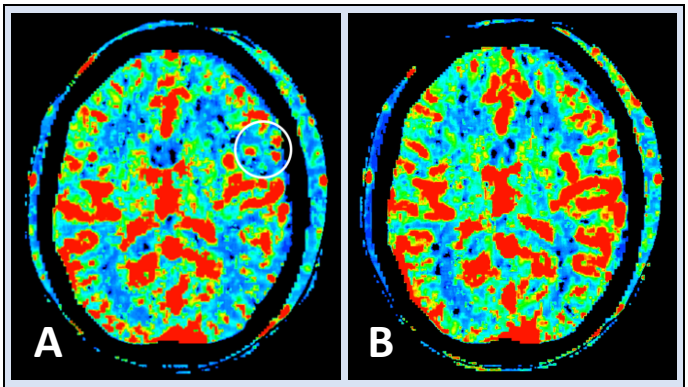

Figure 1. Postoperative perfusion CT findings showing A. altered cerebral blood flow in the frontotemporal region on day (the circle emphasizes the most severe frontal region insufficiency) 5; B. complete resolution on day 9.

Discussion

Post-traumatic vasospasm in our patient developed on the 5th day after the surgery for ASDH, leading to the neurological worsening and confirmed with 3D-CTA. Usual vasospasm treatment protocol led to the CT and symptoms resolution after 4 days.

Cerebral vasospasm is known to occur after head injury; reported incidence reaches up to 63%, however, the screening for PTV is not routine in these patients. Originally, angiographic studies reported PTV rates between 5% and 18.6%, with the introduction of transcranial Doppler sonography and improved imaging modalities, the rates of PTV have increased ranging from 27% to 63% [5].

The origin of PTV remains unknown. Presence of a subarachnoid clot, especially higher Fischer score SAH has been reported to be a major contributing factor in the development of PTV [6], but additional presence of intracerebral [7], subdural and epidural hematomas, SAH [8] are also thought to be causative. However, distinct feature in our patient is occurrence after acute, purely subdural hematoma.

In a wartime cohort, vasospasm was associated with the presence of pseudoaneurysm, hemorrhage, the number of lobes injured, contributing to the injury severity, and impacting the mortality [9, 10]. In our patient, there was no pseudoaneurysm, however, the origin of bleeding was a cortical artery, which may have induced pathophysiological cascade.

The knowledge on the pathophysiology of the PTV development is scarce, and it is most likely a combination of interrelated mechanisms following the SAH, including the altered vascular response to nitric oxide, impact of hemoglobin and its degradation products, inflammation, cortical spreading depolarization and microcirculation disturbance [11]. Hypothalamic dysfunction secondary to increased intracranial pressure and blood degradation products of ASDH might have played some role in the development of the vasospasm in our patient by triggering a sympathetic discharge and catecholamine release [12, 13].

The usual timing for the development of vasospasm after aneurysmal SAH is typically 4-14 days [14]. On the other hand, PTV is characterized by the earlier onset, most frequently in the first 3 days following the injury [4], and it usually lasts less than aneurysmal SAH related; rarely more than 6 days [5]. These characteristics were present in our patient, as the vasospasm occurred on 5th day after injury/surgery and lasted for the following 4 days. Clinically, fever on admission [6], and initial poor neurological status with GCS<9 [15] are also found to be a predisposing factor. All of which were not present in the presented case.

Although it was not possible to apply complete vasospasm treatment protocol due to the traumatic origin of the vasospasm, the treatment based on the triple-H therapy was used for the management with a favorable outcome. The reported clinical outcome of PTV is generally good, but may be also related to the bad prognosis and death [16]. Practical perfusion CT and 3D-CTA were helpful to detect this fairly rare condition, and response with the adequate treatment promptly, regardless of the usual dissociation of the PTV and ASDH.

Conclusions

Delayed PTV can develop even without traumatic SAH, after surgery for ASDH in rare cases. Perfusion CT and 3D-CTA is practical and effective method for early detection in suspicious cases, while the novel therapeutic modalities allow for an uneventful PTV resolution.

Disclosures

Conflict of Interest: All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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References

1. Findlay JM, Nisar J, Darsaut T. Cerebral Vasospasm: A Review. Can J Neurol Sci. 2016;43(1):15-32. doi: 10.1017/cjn.2015.288.
2. Baggott CD, Aagaard-Kienitz B. Cerebral vasospasm. Neurosurg Clin N Am. 2014;25(3):497-528. doi: 10.1016/j.nec.2014.04.008.
3. Perrein A, Petry L, Reis A, Baumann A, Mertes P, Audibert G. Cerebral vasospasm after traumatic brain injury: an update. Minerva Anestesiol. 2015;81(11):1219-28. doi:
4. Witten AJ, Ordaz JD, Alentado VJ, Bohnstedt B. Intracranial Vasospasm After Evacuation of Acute Spontaneous Subdural Hematoma. Cureus. 2021;13(5):e15284. doi: 10.7759/cureus.15284.
5. Al-Mufti F, Amuluru K, Changa A, Lander M, Patel N, Wajswol E, et al. Traumatic brain injury and intracranial hemorrhage-induced cerebral vasospasm: a systematic review. Neurosurg Focus. 2017;43(5):E14. doi: 10.3171/2017.8.FOCUS17431.

6. Shahlaie K, Keachie K, Hutchins IM, Rudisill N, Madden LK, Smith KA, et al. Risk factors for posttraumatic vasospasm. J Neurosurg. 2011;115(3):602-11. doi: 10.3171/2011.5.JNS101667.
7. Oertel M, Boscardin WJ, Obrist WD, Glenn TC, McArthur DL, Gravori T, et al. Posttraumatic vasospasm: the epidemiology, severity, and time course of an underestimated phenomenon: a prospective study performed in 299 patients. J Neurosurg. 2005;103(5):812-24. doi: 10.3171/jns.2005.103.5.0812.
8. Zubkov AY, Lewis AI, Raila FA, Zhang J, Parent AD. Risk factors for the development of post-traumatic cerebral vasospasm. Surg Neurol. 2000;53(2):126-30. doi: 10.1016/s0090-3019(99)00178-0.
9. Armonda RA, Bell RS, Vo AH, Ling G, DeGraba TJ, Crandall B, et al. Wartime traumatic cerebral vasospasm: recent review of combat casualties. Neurosurgery. 2006;59(6):1215-25; discussion 25. doi: 10.1227/01.NEU.0000249190.46033.94.
10. Vitošević F, Milošević-Medenica S. Traumatic intracranial aneurysms associated with traffic accidents and endovascular management options. Serbian J Neurosurg. 2022;1(1):27-32. doi: 10.55005/sjns.v1i1.5.
11. Chugh C, Agarwal H. Cerebral vasospasm and delayed cerebral ischemia: Review of literature and the management approach. Neurol India. 2019;67(1):185-200. doi: 10.4103/0028-3886.253627.
12. Wilkins RH. Cerebral vasospasm in conditions other than subarachnoid hemorrhage. Neurosurg Clin N Am. 1990;1(2):329-34. doi:
13. Wilson JL, Feild JR. The production of intracranial vascular spasm by hypothalamic extract. J Neurosurg. 1974;40(4):473-9. doi: 10.3171/jns.1974.40.4.0473.
14. Bracard S, Schmitt E. Vasospasm and delayed consequences. Interv Neuroradiol. 2008;14 Suppl 1(Suppl 1):17-22. doi: 10.1177/15910199080140S105.
15. Al-Mufti F, Amuluru K, Lander M, Mathew M, El-Ghanem M, Nuoman R, et al. Low Glasgow Coma Score in Traumatic Intracranial Hemorrhage Predicts Development of Cerebral Vasospasm. World Neurosurg. 2018;120:e68-e71. doi: 10.1016/j.wneu.2018.07.143.
16. Armin SS, Colohan AR, Zhang JH. Vasospasm in traumatic brain injury. Acta Neurochir Suppl. 2008;104(13):421-5. doi: 10.1007/978-3-211-75718-5.